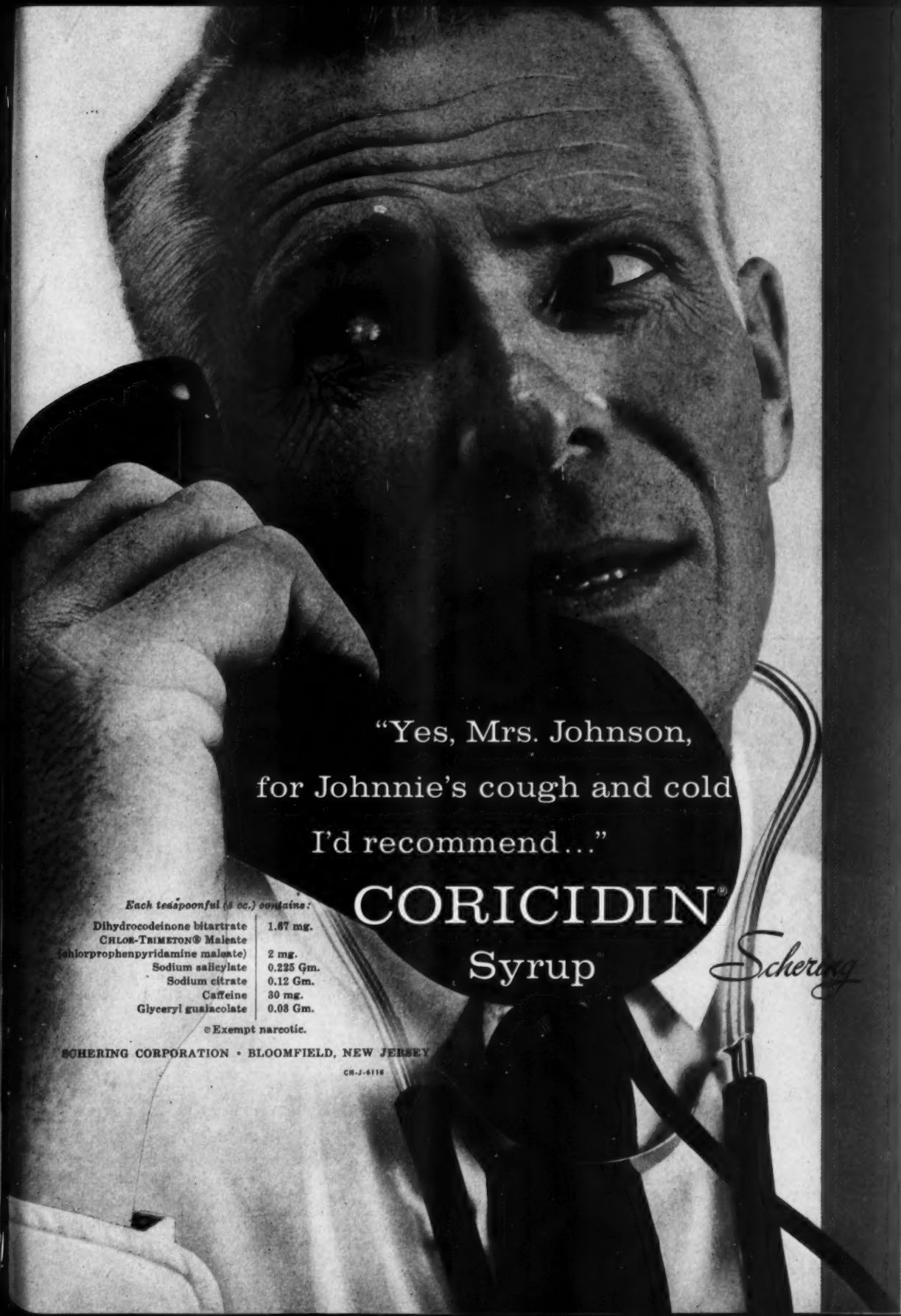
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Contents

Orthopaedic Surgery Notes — Some Comments on the "Spectator Letter" (August 1958) by Dr. Duncan C. McKeever, Houston ..... Page 23
By W. Compere Basom, M.D., El Paso, Orthopaedic Editor

Dermatological Group Announces Essay Contest ..... Page 24

Traumatic Rupture of The Diaphragm with Intrapericardial Herniation of Abdominal Viscera ..... Page 25
By Ralph S. Clayton, M.D., El Paso

Clinical Aspects of The Protein Bound Iodine Determination ..... Page 29
By Jack G. Postlewaite, M.D., El Paso

American College of Surgeons to Meet in Houston ..... Page 34
Arizona Cancer Society to Meet in Phoenix ..... Page 34

Coming Meetings ..... Page 34

Atomic Energy Expert Addresses Southern New Mexico Clinical Meeting, Artesia, November 22 and 23 ..... Page 35

Dr. Johnson of Santa Ana Heads Southwest OB-GYN Society ..... Page 36

Photographic Report of the Southwest OB-GYN Society Annual Meeting in Phoenix ..... Page 37

Dr. Branch Craige New President of El Paso County Medical Society ..... Page 41

Doctors Have Wives ..... Page 42
By Jean M. Craige, El Paso

Aphorisms and Memorabilia — Miscellaneous Truths and Concepts ..... Page 44
Edited by Andrew M. Babey, M.D., Las Cruces, N. M.

Current Therapy — Electrolyte Disturbances in the Treatment of Chronic Heart Failure ..... Page 45
By M. Nathan Kleban, M.D., El Paso

Monthly Clinical Pathological Conference of El Paso General Hospital ..... Page 53
Frederick P. Bornstein, M.D., Editor
Case presentation by Dr. William R. Gaddis

Some Thoughts About Clinical-Pathological Conferences ..... Page 56

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SOUTHWESTERN MEDICINE

VOL. XL JANUARY, 1959 No 1

W. COMPERE BASOM, M.D., *El Paso, Orthopaedic Editor*

ORTHOPAEDIC SURGERY NOTES

Some Comments on the "Spectator Letter" (August 1958) By Dr. Duncan C. McKeever, Houston

Dr. Duncan McKeever has recently written a most interesting spectator letter. Incidentally, "The Spectators," an orthpaedic surgery correspondence group, write letters containing very useful information.

Dr. McKeever, who is the President of the American Fracture Association, brought out a most interesting point: that is the problem of designing and producing a new apparatus for orthopaedic surgery. For instance, in the patella and tibial plateau prosthesis the time and the expense of developing are entirely the burden of the surgeon. The expense of a moving picture to bring the device to the attention of the orthopaedic surgeons in this country was approximately $3,000. The steps in the development of the technique and superb know-how for casting and producing the metallic implants, of course, are borne by the instrument manufacturing company. Dr. McKeever also commented on the quality of work in opinions as given by a group in a questionnaire.

I would like to quote from Dr. McKeever's spectator letter directly from his comments concerning a three-day conference on automobile safety and automobile crash injuries at Ann Arbor, Mich. This was connected with the Medical Education for National Defense Program and was attended by representatives of all the medical schools.

"The most important information obtained at this meeting was that statistically you are about 3½ times as safe from accidental death or severe injury if you have a seat belt on you when you have an accident. Having rolled a station wagon end over end and survived I subscribe to this. For a long time now I have thought that anybody who smokes cigarettes is a damn fool and I am now forced to include people who do not routinely use safety belts in their car in the same category," the letter says.

A paste made of ground up cultured calf bone called Osteogen will shortly be on the market through the Squibb Company. Dr. McKeever has been using this for four years now and he states it is the best bone hemostatic he has ever used. It will instantly stop all bleeding at the donor sites in the ilium and anywhere else you care to use it.

On the subject of cellophane, Dr. McKeever now feels it is preferable to nylon. It cannot be sewed in place and it is more difficult to handle, but it works better. If it fragments or wads up into small balls in the corner of a joint it doesn't seem to hurt anything. This is information gained

from 18 years of use of cellophane and nylon.

In a report of tibial plateau prostheses 26 knee joints are reported. Some of these are over six years old, some of them have one plateau, some of them have both. All but one have a patellar prosthesis. One knee had to have the prosthesis removed because of infection. All of the others are functioning satisfactorily and the patients are walking on them without crutches. In some of the knees an arthrodesis could have been indicated. The prostheses apparently are successful and an advantage to the patient in a non-infected knee. Four successful elbow prostheses with replacement of the entire lower end of the humerus have been far better than a stiff elbow or a flail elbow.

Arthrodesis of the ankle and subastragalar joint with proper foot position give knee stability in extension and weight bearing and enable patients to get by without braces. The hip prosthesis continues to function satisfactorily. It is not subject to torque.

Dr. McKeever recommends that we read the book "Atlas Shrugged". In fact he says "to any of you who are faint of heart, I'll dare you to read it."

I enjoyed reading Dr. McKeever's copy of "Hospitals Are For Sick People" in which the patient has so much disturbance that he had to go home to rest and recover. Also the overemphasis of paper work in preference to emphasis of treatment of patients is a problem which all of us face.

I enjoyed the verse quoted as follows:

"Adventure on, for from the littlest clue
Has come whatever worth man ever knew.
The next to lighten all men, may be you!"

Dermatological Group Announces Essay Contest

The Los Angeles Dermatological Society has established a Nelson Paul Anderson Memorial Fund which offers a $500 cash award annually for a minimum of five years, for a winning essay to be read by the essayist at the annual meeting of the Pacific Dermatological Association. The Pacific Dermatological Association will pay the basic expenses (transportation, room and board) of the winning essayist to, at and from the meeting.

The essays eligible for the contest shall report original work, not previously published, formally presented or previously prize-winning, relative to some fundamental aspect of dermatology.

The essays shall be judged on the following considerations: a. originality of ideas, b. potential importance of the work, c. experimental methods and use of controls, d. evaluation of results, e. clarity of presentation.

The essay shall be submitted under a "nom de plume" with no information anywhere in the paper which might lead to recognition by the judges of the institution or clinic at which the work was done. The essay with "nom de plume" shall be accompanied by a plain sealed envelope enclosing the name and address of the author; this envelope shall not be opened until the judging is complete.

The contest shall be open only to physicians engaged in the study or practice of dermatology, who are working within the geographical limits encompassed by the Pacific Dermatological Association.

The essays will be judged by a committee of five judges, appointed by the Executive Council of the Pacific Dermatological Association, and all entries should be in the hands of Louis H. Winer, M.D., 9915 Santa Monica Blvd., Beverly Hills, Calif., in quintuplicate (all copies clearly legible, of course) before May 1, 1959. The winner will be notified by Aug. 1, 1959; the 1959 meeting of the Pacific Dermatological Association will be held at the La Playa Hotel, Carmel-by-the-Sea, Calif., Sept. 9-13, 1959.

ORIGINAL ARTICLES

Traumatic Rupture of The Diaphragm with Intrapericardial Herniation of Abdominal Viscera

By Ralph S. Clayton, M.D.,* *El Paso*

We are unable to locate any report of isolated rupture of the central tendon of the diaphragm with intrapericardial herniation of abdominal viscera due to non-penetrating trauma to the chest or abdomen. It is recognized that early diagnosis of rupture of the diaphragm is important in order that complications may be prevented and surgical repair effected at the proper time. Because of the increasing incidence of high velocity and crushing type injuries to the chest and abdomen in automobile and aircraft accidents, we believe this lesion should be brought to the attention of radiologists and surgeons.

Case I

Mr. A.C.J., age 41, was hospitalized April 8, 1950, for injuries received that day in an automobile accident. He had sustained severe injuries to the chest and pelvis. A radiograph of the chest on admission showed a small amount of fluid in the left pleural space. Fractures of the left 2nd through the 10th ribs and of the right 2nd, 3rd and 4th ribs were demonstrated. There were multiple fractures of the pelvis.

The left pleural fluid increased steadily in amount during the week following the accident, and was interpreted as hemothorax. Twelve days after admission, radiographs of the chest showed the splenic flexure of the colon above the diaphragm in the lower portion of the left side of the chest anterior to the heart. (Figs. 1 & 2) Barium studies confirmed these findings. (Fig. 3) About three weeks after the accident a thoracotomy was performed to evacuate the left hemothorax and to repair the diaphragmatic hernia. The hemothorax was evacuated but exploration of the left pleural space failed to reveal any abdominal viscera. Several of the rib fractures were stabilized with bone pegs.

Five days after the thoracotomy, radiographs of the chest again demonstrated the splenic flexure of the colon to be above the diaphragm in the same location as before. Pleural fluid reaccumulated rapidly in the left pleural space. Temperature elevation indicated that an empyema was developing, and frequent aspirations were performed.

Because of the persisting findings of diaphragmatic hernia, abdominal exploration was

Fig. 1 (Case 1)

Antero-posterior supine radiograph of chest shows splenic flexure of colon above diaphragm. Barium is in fundus of stomach. Note pleural fluid on left.

*Formerly Director, Department of Radiology, Parkland Memorial Hospital, and Clinical Associate Professor of Radiology, Southwestern Medical School, Dallas, Texas.

performed five weeks after admission. A 12 cm. tear of the central tendon of the diaphragm and pericardium was found which permitted the omentum and splenic flexure of the colon to herniate into the pericardial space. Numerous adhesions to the pericardium were present. The tear was about 1 inch from the attachment of the diaphragm to the anterior chest wall. The defect was closed without tension by overlapping the

Fig. 2 (Case I)

Lateral radiograph of chest shows colon above diaphragm in anterior mediastinum.

torn edges of the diaphragm with interrupted mattress sutures of No. 3 braided silk. The abdomen was closed in layers. To evacuate the empyema of the left pleural space, a pleural drainage tube was inserted and connected to a waterseal system.

The further course of this patient was uneventful.

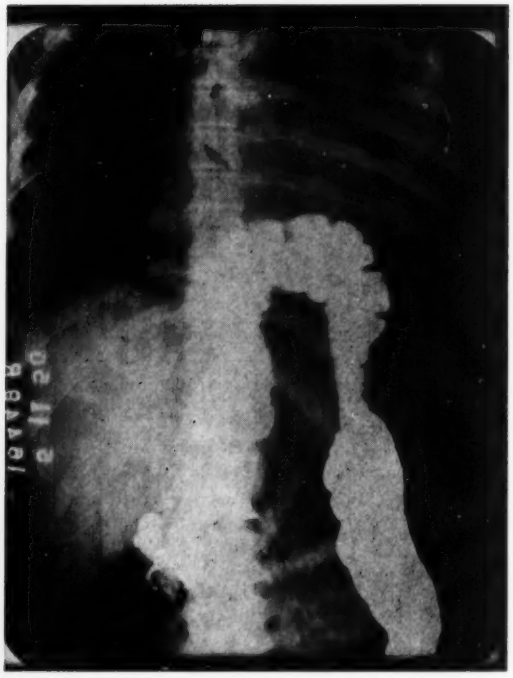

Fig. 3 (Case I)

Splenic flexure of colon is opacified by means of barium enema.

Case II

Mrs. G.G., age 32, was hospitalized on December 24, 1950, for injuries incurred in an automobile accident a few minutes earlier. The patient was unconscious immediately after the accident, but regained consciousness for a short time and complained of pain in the left chest anteriorly. She was restless and irrational on admission. The working diagnosis was head injury.

Radiographs of the skull did not reveal any bony injuries. A radiograph of the chest revealed fractures of the left clavicle, the right 2nd rib and the left 1st rib. The diagnosis of traumatic diaphragmatic hernia was made from this emergency anteroposterior supine chest film. (Fig. 4)

Her condition remained critical for the next 10 days because of the brain injuries. By this time, further examination of the chest injuries became possible. Fluoroscopic and radiographic studies revealed herniation of most of the stomach through the central tendon of the diaphragm, but specific localization of the herniated viscus inside

the pericardial space was not made at that time. (Figs. 5 & 6)

On January 4, 1951, a thoracotomy was performed through the bed of the left 9th rib. No evidence of diaphragmatic hernia could be found in the pleural space. However, because the surgeon was the same individual who performed the initial surgery in Case I, the pericardial sac was opened and the hernia found. An incision was then made through the left leaf of the diaphragm and the hernia observed from below.

A portion of stomach and omentum comprised the contents of the hernia. They were returned to the abdomen without difficulty. The diaphragmatic defect was large enough to admit the surgeon's hand with ease. Interrupted mattress sutures of braided silk were used to close the defect in the usual overlapping fashion. A second small tear was found in the diaphragm, without any hernia, communicating with the left pleural space. This was closed with interrupted cotton sutures.

The diaphragmatic surgical incision was closed by the usual technic. An intercostal drainage tube was inserted into the pleural space and connected to a waterseal system. The lung was re-expanded and the chest was closed in the usual manner.

The subsequent course of this patient was uneventful.

Fig. 5 (Case II)
Barium proves live shadow of Fig. 4 to be stomach.

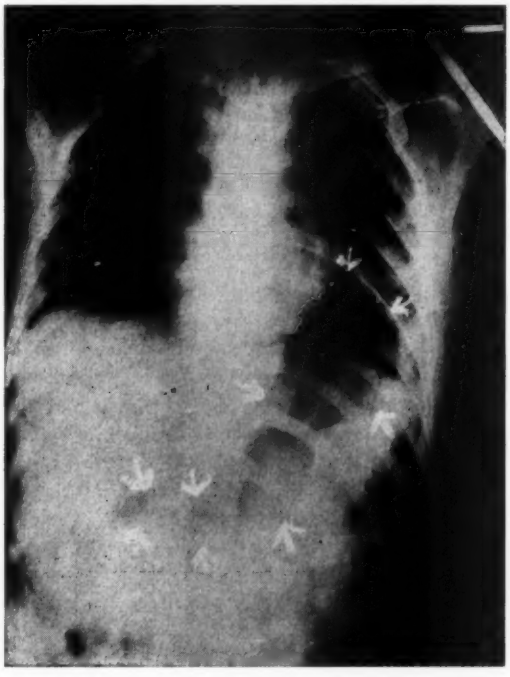

Fig. 4 (Case II)
Antero-posterior supine radiograph of chest showing air shadow above diaphragm and intimately related to the heart.

Discussion

In retrospect, in Case I, the location of the herniated viscus directly anterior to the cardiac shadow would have suggested the correct diagnosis if intrapericardial hernia had been considered. No observations were recorded concerning the effect of heart action on the barium filled colon, (see Case II below).

The radiologic findings in Case II were unique in several respects. These findings may be tabulated as follows:

(1) The supine anteroposterior radiograph of the chest showed the apex of the heart to be displaced upward and to the right by gas.

(2) This gas shadow was limited laterally to the left by a soft tissue line which was the shape of the pericardium and was in practically the normal position of this structure.

(3) The gas shadow was continuous through the diaphragm with air in the stomach within the abdomen, suggesting herniation of the stomach.

(4) The right heart border was in normal re-

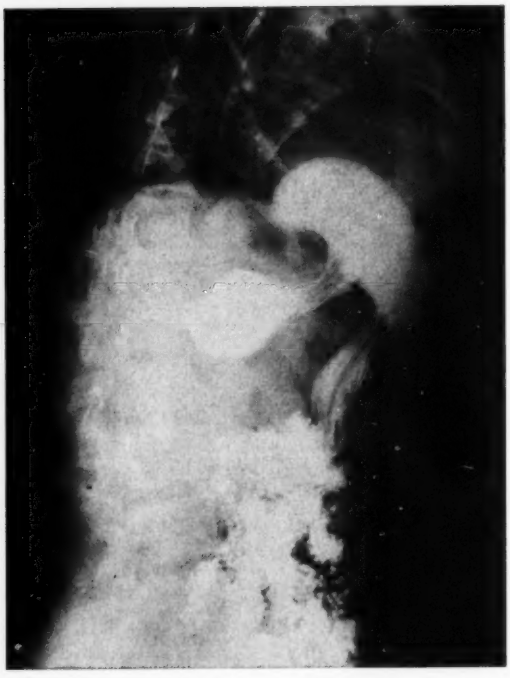

Fig. 6 (Case II)

Left posterior oblique projection of barium filled stomach showing it to be located in the anterior mediastinum.

lation to the spine, whereas it is usually displaced to the right in the intrapleural herniation of abdominal viscera into the left hemithorax.

(5) The shadow normally cast by the left half of the central tendon of the diaphragm was absent. This was later shown to be due to the actual tear through the diaphragm and pericardium.

(6) The fluoroscopic findings were quite striking but were not correctly interpreted pre-operatively. The cardiac border near the apex was much more clearly visible than usual. The heart border in the same area could not be separated from the gas shadow by rotation of the patient, which should have suggested intrapericardial location of the gas. Barium flowed normally down the esophagus, through the diaphragm, and then coursed upward around the apex of the heart in the gas previously described.

The wild motion imparted to the barium by the heart was commented upon by the fluoroscopist, and provided additional evidence of the intrapericardial location of the stomach. This motion was much more vigorous than when the stomach is in the pleural cavity. These observations were made during fluoroscopy but their significance was not appreciated at that time. Consequently, the pre-operative radiologic diagnosis was simply traumatic rupture of the diaphragm with intrapleural herniation of the stomach.

The fluoroscopic findings described in Case II suggest that fluoroscopy should be performed whenever the herniated viscera lie close to the heart.

The surgical approach may be planned to better advantage if the precise location of the hernia is known prior to operation.

These cases illustrate the fact that intrapericardial hernia may not be found at thoracotomy unless the possibility is kept in mind and the pericardium opened when the pleural space does not contain the herniated viscera demonstrated by x-ray.

Conclusions

The proper planning of the surgical repair of traumatic rupture of the diaphragm is dependent on accurate radiologic diagnosis. If radiologists and surgeons are aware of the entity of intrapericardial hernia as a result of non-penetrating injury, and are familiar with the differences between the findings in intrapericardial herniation and intrapleural herniation, an exact pre-operative diagnosis can be made.

We feel that the following findings should be sufficient for a definitive diagnosis: (1) The location of the abdominal viscus in intimate relation with the heart. (2) The striking effect of heart action on the contrast filled viscus. (3) Other ordinary findings of diaphragmatic rupture such as loss of diaphragm contrast and abnormal tissue shadows above the diaphragm, with or without displacement of the heart shadow itself, in a patient who has sustained severe trauma.

Summary

1. Two cases of traumatic rupture of the diaphragm with intrapericardial hernia, due to non-penetrating trauma, are reported.

2. Radiologists and surgeons should be aware of the possibility of this lesion.

3. Radiologic criteria and surgical considerations are described.

1501 Arizona Ave.

(The surgeon in both cases was Dr. Donald L. Paulson, Clinical Associate Professor of Surgery, Southwestern Medical School, Dallas, Texas.)

Clinical Aspects of The Protein Bound Iodine Determination*

By Jack C. Postlewaite, M.D., El Paso

The problem of thyroid hormone and metabolism is a phase of the whole question of iodine metabolism. It is only in the manufacture of the thyroid hormone that the body uses iodine. That it uses iodine is the most unique thing about the thyroid. The presence of iodine in the molecule of its hormone is of special significance.

Harington and Barger[1].* produced a compound like thyroxine, but without its four atoms of iodine. Physiologically this dexiodothyronine was metabolically and physiologically inert. However, when two atoms of iodine were incorporated in the molecule, slight physiologic activity appeared, and when four iodine molecules were placed correctly the hormone attained natural potency. Iodine ingested beyond the need of thyroid hormone manufactured is very promptly eliminated.

Iodides Useful

Iodides are useful for their pharmacologic action, for example as expectorants. Such iodine exists and circulates in the body as inorganic salt and plays no part in metabolism or bodily economy. Its presence in the body as inorganic salt plays no part in the function of the thyroid. The iodine content of the blood is in two parts; as passive iodine, and as circulation hormone. The same is true of the iodine content of other tissues.

The former fluctuates widely with iodine intake, the latter is related to the function of the thyroid. The buffering effect of the thyroid storehouse is gone. A thyroid ingestion of a quantity of iodine is followed by a great rise in blood iodine, and urinary iodine, than would be the case in the organism in possession of its thyroid.

Multiple Channels

The absorption of iodine is through multiple channels. The usual route is through the alimentary canal. It may likewise be absorbed through the skin, the lungs, or by any absorption membrane.

Rate of absorption varies with the solubilities of the compounds in which the iodine occurs. Thyroxine polypeptide by virtue of its solubility is absorbed almost entirely from the alimentary tract, whereas thyroxine, which is less soluble, is absorbed to a much less extent (from 10 to 20 percent).

The excretion of iodine is through the kidneys, the sweat glands, the bile and the salivary glands. The daily amount in the urine is dependent on the intake. In thyrotoxicosis the urinary excretion is raised because of increased manufacture of hormone and depletion of storage of iodine for example.

Men using noniodized salt excrete 9 gammas and those using iodized salt, 121 gammas. Strenuous exercise doubles the iodine output and also the day urine contains about twice as much as the night.

No Evidence

There is no evidence to indicate that a renal threshold for iodine plays any role in the regulation of the body's store of iodine. Endogenous iodine metabolism is a function of the thyroid. The physiologic activity of the whole thyroid gland is determined by its total organic iodine content.

The daily requirement of hormone can be estimated by discovering what quantity must be given to thyroid individual in order to keep him in a normal condition over a long period of time. Plummer[1]* has calculated that in terms of thyroxine the daily requirement is in the neighborhood of 0.5 to 1 mg. or 0.3 to 0.6 mg of iodine. A fullbloom myxedema can be maintained on 100 mg. of dried thyroid or 0.22 mg of iodine.

The body needs less than 0.33 mg. of thyroxine which is the amount destroyed in the body or excreted in the body.

It is interesting to note that the iodine requirements of the body for thyroxine manufacture is vastly greater than the apparent absolute iodine requirement of the body as a whole.

Body Economy

The body has an economy with iodine and utilizes it over and over again in hormone synthesis. Of the iodine derived from the daily breakdown of thyroid hormone only a fraction is excreted. The thyroid requirements of athyroid children is greater than athyroid adults.

The normal thyroid of the human weighs 20 grams wet, and contains 0.2 percent of iodine or

*Presented before the Texas Society of Medical Technologists.

a total of 8 mg. of iodine. Of this 4 mg is in the form of thyroxine iodine and the balance is diiodotyrosine iodine.

Extra thyroid iodine is no more than 10 to 14 mg. of thyroxine of which 6 to 7 mg. is in the form of thyroxine iodine. There is 15.0 mg. of iodine in the rest of the body in combination other than as thyroxine.

The liver is an organ of destruction of excesses of thyroid in the body, and not by simple excretion. The iodine concentration in the blood was shown by Gley, et. all, [1]* in 1900 to be a constituent of normal blood. In 1925, Viel[1] showed that in thyrotoxicosis, total blood iodine was increased and in hypothyroidism decreased.

Fractionating Blood Iodine

In 1929, Lunde devised a method for fractionating the total blood iodine into an alcohol soluble and an alcohol insoluble fraction. Salter separated plasma iodine and felt that the concentration of protein bound iodine in human plasma was a reliable index of net thyroid function. He regarded this fraction as circulation hormonal iodine.

Studies made with radioactive iodine indicate that the thyrotropic hormone will promote the release and uptake of thyroid iodine.

The first effects of TSH on iodine metabolism in the thyroid would appear to be the purging of the thyroid of its stored iodine. The loss of iodine would appear to precede the hypertrophy of the thyroid cell which occurs when TSH is administered.

It is suggested that TSH is activated to some extent as proteolytic enzyme present in the thyroid colloid.

Although the mode of action of TSH on the thyroid cells in unknown, Rawson[1] has suggested that in exerting its influence in the thyroid, the thyroid stimulating hormone is inactivated.

Other Tissues

The only other tissues which caused any inactivation of TSH invitro were the thymus and lymph nodes. The involvement of the thyroid stimulating hormone in disease has not been so well delineated.

It has been observed that the urine of patients with untreated Graves' disease contains no active TSH whereas after treatment of the patients with thyrotropic substances have been observed in the urine. Thyroid tissue of patients with Graves' disease, with invitro techniques, was found to inactivate more TSH than normal.

The interesting developments in thyroid studies are with triiodothyrone investigation. It exerts a much more immediate calorigenic effect and thyrtropin inhibitory effect than thyroxin and is found in smaller quantity in the body.

It, like thyroxin, is active by mouth. Since l-thyroxin alleviated myxedema and occurs in plasma, it is generally accepted as the thyroid hormone.

Several Paradoxes

However, several paradoxes exist: (1) dessiccated thyroid has greater calorignic activity than accounted for by its l-thyroxin content; (2) unlike most hormones, delay in specific effects is observed after exhibition, and (3) in some athyrotic patients receiving l-thyroxin, the serum protein-bound iodine level is higher than anticipated by their clinical and metabolic status.[2]

Preliminary studies with the newly discovered l-triiodothyronine indicates that it produces an immediate metabolic effect 5-10 times that of equivalent amounts of l-thyroxin. The remarkable activity of l-thyronine affords explanation of these paradoxes.

Within six hours of administration of 0.5 to 1.0 mg of l-triiodothyronine in a single subcuteneous dose, the pulse rate and body temperature progressively increased, reaching a maximum on the third day. Weight loss and diuresis also occurred.

The BMR increased promptly. l-triiodothryonine accelerated urinary creatin excretion; and nitrogen and phosphorus diureses develops, resulting in negative nitrogen and phosphorus balances. After administration of equivalent amounts of l-thyroxin, significant but minimal metabolic changes occurred.

Iodine Levels

Serum protein-bound iodine levels increased after administration of l-triiodothyronine, but often remained in ranges found in hypothyroidism when the patients were metabolically euthyroid; yet l-thyroxin increases the value to levels found in euthyroidism, although the patients remained hypothyroid.

Serum cholesterol levels decrease after administration of either l-thyroxin or l-triiodothyronine; this decrement does not bear quantitive relationship to the degree of metabolic change observed. The electrocardiograms reveal more rapid reversion to normal after l-triiodothyronine than after l-thyroxin.

After l-triiodothyronine, ballistocardiograms first show acute deterioration in form but subsequent improvement. l-triiodothyronine alleviates myxedema more rapidly and with smaller doses than l-thyroxin and may represent the functional constituent of the thyroid hormone.

Thyroid Ingestion

The ingestion of 6-180 mg. of thyroid hormone depresses the activity of the thyroid gland in 90

percent of individuals. In cessation of ingestion of thyroid substances by normal individuals the basal metabolic rate falls to below pre treatment control levels.

In myxedematous patients the BMR falls to pre treatment levels and not below. A fall of serum protein-bound iodine to below normal levels was noted in euthyroid subjects after discontinuance of thyroid medication.

Recently Greer[3*]. found that the uptake of radioactive iodine by the thyroid gland of normal persons was markedly inhibited by the ingestion of dessicated thyroid. It returned to normal within four weeks.

Thyroxine if not truly the active thyroid hormone is clearly the principal iodine-containing compound released by the thyroid gland, and ordinarily comprises the largest fraction of the plasma's protein bound iodine.

Many investigators [3] have, therefore attempted to access thyroidal function by measuring the concentration of protein bound iodine^{131} in the plasma following the administration of radioactive iodine.

The PBI or the total radioactivity in the plasma determined several days after the administration of the tracer reflects the radioactivity of circulating thyroxine, except patient who have received large doses of I^{131}.

Hormonal Iodine

It has been shown that the hormonal iodine which is extractable from the serum with butanol and not re-extractable with alkali is composed largely of thyroxine.

Its concentration in serum differs significantly from that of the protein bound iodine.

Contamination of the sample of serum with iodine has caused little trouble in the laboratory study of PBI. A more subtle error in the serum PBI is from patients who have been given mercurial diuretics. These values are consequently rendered low.

Patients who have ingested large quantities of iodine or ingested iodine containing radio-opaque material for example: Graham Cole x-ray study of the gallbladder, intravenous pyelography, or bronchography are found to have high values for PBI. A careful history should be taken to avoid the possibility of introducing error from such sources.

Other Drugs

Neo-penil.[4] (nenethamate hydriodide) has been shown to elevate the PBI for one week beyond the discontinuation of therapy. Other drugs which may influence the concentration of protein-bound iodine include the iodides, iodothiouracil, and iodine containing amebicides[5].

Even barium mixtures used in gastrointestinal studies may contain enough iodine to affect blood levels.

The concentration of serum precipitable iodine (PBI) increases in men and women during the administration of estrogen. A leveling off in the increments is usually observed by the third or fourth week. Normal functioning of the pituitary and thyroid are necessary for the reaction to occur.

The PBI falls to control values when the estrogen is discontinued. Data are present to indicate that the increments in PBI are due to an increase in activity of the thyroid, at least for a period of time, and are not due to an altered rate of disposal of thyroid hormone. Elevated PBI levels are found in normal pregnancy[6].

Two Rare Cases

Two rare cases of elevated PBI in myxedema have been reported by P. S. Blom, M.D. of Leiden, Holland[2]. After surgical treatment only very small remnants of thyroid gland tissue are left functioning, and these remnants (under increasing TSH stimulation) continue the properties of the total gland in hyperthyroidism. An essential feature of these glands is the rapid turn over rate of iodine with only slight storage of organically bound iodine.

The total iodine content of the gland in hyperthyroidism is considerably less than in a normal gland, and iodine collected by the gland will be quickly transferred to the blood after being transformed into thyroxine.

Poor Correlation

In several patients who had undergone virtually complete thyroidectomy, however, the concentration of PBI did not correlate well with the clinical status of the patient. An explanation for this discrepancy was sought in an analysis of the factors determining the concentration of PBI in the plasma.

The principal and perhaps only source of plasma's thyroxine appears to be the pool of hormonal iodine within the thyroid gland; therefore, the relationship for a product substance and its immediate precursor can be applied to the relationship existing between thyroxine in the plasma and in the thyroid gland.

Analysis of correlative studies reveals that the concentration of PBI in the serum is dependent on the initial uptake of I^{131}, the rate of synthesis

of thyroid hormone, and the amount of hormone stored within the thyroid gland. The 72 hour concentration of PBI correlates well with the clinical state except in non thyrotoxic patients in whom the quantity of hormone stored within the gland is markedly decreased.

With Albumin Fraction

Iodine appears first with the albumin fraction of the blood where it is associated with the slower moving component electrophoretically. It is later found in a zone of low protein concentration just ahead of alpha-2 globulin. This behavior was observed in the serum from both euthyroid and hyperthyroid subjects.

However, in hyperthyroid subjects the tracer iodine concentrates faster, attained higher levels, and declined more rapidly in the alpha globulin zone.

This suggests that the hormonal iodine may be associated with this specific protein. The exact nature of this iodine compound is unsettled although the presence of thyroxin and more recently 3:5:3′ L triiodothyroxine has been demonstrated in whole serum.

Increase In Heat

Thirty years ago Marine[1] noted that the adrenal insufficiency in rabbits led to an increase in heat production and respiratory exchange and to the appearance of a symptom complex resembling exophthalmic goiter.

Marine[1,7] subsequently reviewed the evidence pointing to the importance of a primary adrenogonacal insufficiency as a possible fundamental cause of Graves disease. The occurrence of Graves disease following roentgen ray damage to the adrenal cortex in 2 cases provided a direct clinical example in support of this concept.

The pathologic changes observed in patients who have died of thyrotoxicosis enlargement of the thymus, diffuse lymphoid hyperplasia and small adrenal cortices have been cited as evidence suggestive of adrenal cortical insufficiency.

The endocrine antithesis to the hyperthyroid hypoadrenocortical state is well demonstrated by the frequent occurrence of an atrophied thyroid gland in Cushing's syndrome.

Dry Skin

The high concentration of serum cholestrol, the dry skin, and the low BMR frequently present in this disease are clinically suggestive of hypothyroidism.

The availability of ACTH and cortisone has stimulated further experimental inquiry into the nature of thyroid adrenal relationships. In Addison's disease, the thyroid depression following initial stimulation produced by cortisone therapy of more than four days duration led to a trial of cortisone therapy in hyperthyroidism, with variable results.

In 5 to 23 days of therapy 3 improved and 6 noted decrease in the size of the organ. They noted a fall in the I^{131} accumulation gradient although the 24 hour iodine uptake did not change.

ACTH apparently retarded the accumulation of L^{131}. In 8 of 13 patients the serum protein bound iodine levels fell but the BMR did not decrease until the changes in accumulation gradient and in the serum protein bound iodine level became marked[8].

Little Close Correlation

In general close correlation of the iodine accumulation function with the BMR and the serum cholesterol concentration was not observed. It has been pointed out that significant alteration in these latter measurements may not be observed during a short course of cortisone therapy.

In almost all cases the return to pre-treatment levels was manifested within a few days to a week following discontinuation of the drug or reduction of dosage to 50 mgs. or less a day.

Under continued therapy the thyroid inhibition became progressively more marked but renal clearances tended to return towards pre-treatment levels in some cases.

It appears that the effect of cortisone therapy depressed the radioiodine accumulation to a striking degree and is usually associated with a fall in the level of the serum PBI.

Appears Early

It is apparent that this effect appears early after the onset of cortisone administration and that the time required for recover if cortisone is stopped of this function after cessation of therapy may be very short 1-2 days if cortisone is given by mouth, but prolonged 2-7 weeks if the intramuscular routine is used.

Another point of interest is the failure of a sharp rise in PBI level, noted in several patients precipitated into thyroid crisis or storm.

The behavior of the PBI has been studied after x-ray, surgical, and radioactive iodine therapy for toxic goiter and similar increases have been noted occasionally without crisis.

This agrees with the clinical impression that thyroid crisis is not the consequence of an exacerbation of the disease but rather is the end result of long standing and neglected disease regardless of the degree of thyrotoxicosis.

Basal Metabolic Rate

Elevation of the basal metabolic rate is subject to error in the evaluation of suspected hyperthyroidism. Cerebral stimulation, increased metabolism of malignant cells, pregnancy, pheochromocytoma, hemodynamics, factor alterations of congestive heart failure, and other extra thyroidal states affect the metabolic rate.

The resultant BMR is less in error than it is in interpretation for it is not a selective test of thyroid hormone, but indicates the end result of many oxidative processes of the body.

Forty-nine patients studied by Meckstroth et. al., had initial diagnosis of hyperthyroidism. Many of the patients had symptoms of toxicity, such as nervousness, weight loss, increased sweating, palpitation and fatigability, and all had an elevated BMR.

Diffuse enlargements of the thyroid predominate in this series where as others showed no thyroid abnormalities.

Effective In Diagnosis

Serum protein-bound iodine levels were effective in diagnosing euthyroidism in 95 percent of the patients, radioactive iodine uptake in 89 percent, serum cholesterol levels in 72 percent, and somnolent metabolic rate in 53 percent.

The true diagnosis in eleven patients was psychoneurosis; six, leukemia; three, diabetes; and five, cardiovascular disease. This indicates that thyroid physiology is so complex that one test will not evaluate the glands functional state.

The four tests can not be compared, since each test is a measure of a different phase of the thyroid metabolism. The RAI uptake determined by a single 24 hour count tells only how much I^{131} is in the gland at that time.

The rate of uptake or the rate of release is not determined. Serial counts are necessary for this information.

However, the RAI is not elevated by the common functional thyroid disorders, nor by organic non-thyroid diseases.

Tracer Studies

The results of tracer studies are disturbed by exogenous iodination and the source of iodination is difficult to locate. The serum PBI is generally accepted to consist of calorigenic substance. The most direct measure of thyroxin formation is found in PBI of the serum.

PBI is not affected by diseases causing extrathyroid hypermetabolism, and is affected by exogenous iodination. The BMR measures total body metabolism and can not differentiate between the etiologic factors in the hypermetabolism.

Only acclimatization to the metabolism procedure by multiple tracings can assure a reliable study. The SMR is determined under anesthesia and is especially valuable in functional disorders.

Many patients with hyperthyroidism are often complicated by nervousness which adds to an already elevated BMR. The low value of 53 percent diagnosis compatibility of the SMR is misleading as the cases of extrathyroid hypermetabolism are not usually organic, such as leukema or diabetes, but rather psychoneuroses.

Upper Limits

The normal SMR value of —7 percent is considered the upper limits of normal. In the 11 psychoneurotic patients, the value was under —6 percent; on eight occasions, 72 percent. The highest SMR was —1 percent. The SMR gives a reproducible value more often than a BMR which fluctuates from day to day. The administration of iodine, thiouracil derivatives, or therapeutic doses of I^{131} does not interfere with this test and is the best single test to follow all classes of therapy for hyperthyroidism[9].

1501 Arizona Ave.

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American College of Surgeons to Meet in Houston

All members of the medical profession are invited to attend a three-day sectional meeting of the American College of Surgeons in Houston, Feb. 2-4, at the Shamrock Hilton Hotel.

Reports on general surgery, a full day's otolaryngology program, a cancer workshop, films, and hospital clinics will comprise the three-day meeting.

The special otolaryngology session on Monday, Feb. 2, will include scientific reports and discussions on problems of current concern. The cancer workshop on Wednesday, Feb. 4, is for medical directors of approved cancer programs in Southwestern United States, and its purpose is to improve the professional and administrative aspects of local cancer programs.

First Program

It is the first program of its type to be conducted in many years, and its success will determine future use of workshops during sectional meetings. Dr. R. Lee Clark, Jr., chairman, Executive Committee of the American College of Surgeons Committee on Cancer, is in charge.

General sessions will include discussions on tumors, varicose veins, preparation of parents for parenthood, cancer, radiation hazards, diseases of the pancreas, gastrointestinal tract bleeding, among other topics.

Dr. H. Prather Saunders, Associate Director of the American College of Surgeons, is in charge of all sectional meetings for the College.

Arizona Cancer Society to Meet in Phoenix

The seventh annual seminar of the Arizona Division, American Cancer Society, will be held Jan. 22-24, at the Paradise Inn in Phoenix.

Speakers and panelists for the scientific program are:

Dr. Alfred Gellhorn, Associate Professor of Medicine and Director of the Institute of Cancer Research, Columbia University; Dr. Warren Bostick, Associate Professor of Pathology, University of California; Dr. Howard Hunt, Director of Tumor Clinic and Professor of Radiology, University of Nebraska.

Dr. Alexander Brunschwig, Professor of Clinical Surgery, Cornell University Medical School; Dr. James W. Kernohan, Professor of Pathology, Mayo Foundation, Rochester, Minn.; Dr. Phillip Hodes, Professor of Radiology, University of Pennsylvania; Dr. Edwin M. Boldrey, Associate Professor and Chairman of Department of Neurosurgery, University of California.

Dr. L. H. Garland, Professor of Radiology, Stanford University; Dr. Richard Overholt, Thoracic Surgeon and Clinical Professor of Surgery, Tufts College, Boston; and Dr. W. A. D. Anderson, Professor of Pathology, University of Florida School of Medicine.

Coming Meetings

Southwestern New Mexico Medical Society, Deming Country Club, Deming, N. Mex., Jan. 15, 1959.

University of Colorado Medical Center, Denver, Colo., Postgraduate Course, General Practice Review, Jan. 19-24, 1959.

American College of Surgeons, Sectional Meeting, Shamrock Hilton Hotel, Houston, Texas, Feb. 2-4, 1959.

University of Texas Postgraduate School of Medicine, Temple Division, Seventh Postgraduate Conference in Medicine and Surgery, sponsored by Scott, Sherwood and Brindley Foundation, Temple, Tex., Mar. 2-4, 1959.

University of Colorado Medical Center, Denver, Colo., Postgraduate Course, Medical Technology, Mar. 16-21, 1959.

Biennial Western Conference on Anesthesiology. Approved for 24 hours Category II Credit, A.A.G.P. Westward Ho Hotel, Phoenix, Apr. 1-4, 1959. For information write to Boyden L. Crouch, M.D., 301 W. McDowell Road, Phoenix, Ariz.

The Arizona Medical Association, 68th Annual Meeting, San Marcos Hotel, Chandler, Ariz., April 29-May 2. Friday, May 1, has been designated as Medical Education Day.

New Mexico Medical Society, annual meeting, Las Cruces, N. Mex., May 5-7, 1959.

Southwestern Medical Association, Annual Meeting, Roswell, N. M., Nov. 5-7, 1959.

MEETINGS

Atomic Energy Expert Addresses Southern New Mexico Clinical Meeting, Artesia, Nov. 22 & 23

ATOM EXPERT—Dr. Gerald W. Johnson, right, Test Division Leader, University of California Radiation Laboratories, was speaker at a banquet in Artesia Nov. 22 held in conjunction with the semi-annual meeting of the New Mexico Medical Society's House of Delegates and the Southern New Mexico Clinical meeting Nov. 23. On the left is Dr. Earl B. Flanagan of Carlsbad. Dr. Johnson's subject was "Research in the Peaceful Use of the Atom" and embraced a discussion of the forthcoming underground atomic blast slated for the salt beds near Carlsbad.

SOUTHERN NEW MEXICO CLINICAL MEETING—Among speakers and guests at the Southern New Mexico Clinical meeting in Artesia Nov. 23 were, left to right, Dr. R. A. Manganaro, Carlsbad, president-elect of the Eddy County Medical Society; Dr. George Richardson, Roswell; Dr. Sol Heinemann, Carlsbad, public relations chairman for the New Mexico Medical Society; Dr. J. J. Clarke, Artesia, a dentist who received his license in New Mexico in 1906 and who has been Secretary of the New Mexico Board of Dental Examiners since 1923 and a member of the Board since 1912; Dr. Albert J. Fisher, Albuquerque; Dr. Louis F. Hamilton, Artesia, moderator; and Dr. Earl B. Flanagan, Carlsbad.

Dr. Johnson

Dr. Johnson of Santa Ana Heads Southwest OB-GYN Society

Dr. Donovan Johnson of Santa Ana, Calif., was elected President of the Southwest Obstetrical and Gynecological Society at its eighth annual meeting in Phoenix November 14 and 15.

Other new officers are Dr. Charles J. Newcomb, Tucson, President-Elect; Dr. John F. Wanless, San Diego, Vice-President; Dr. Zeph B. Campbell, Phoenix, Secretary; and Dr. Raymond J. Jennett, Phoenix, Treasurer.

Elected to the Council were Dr. Louis A. McRae, Jr., Albuquerque, for New Mexico; Dr. Gray E. Carpenter, El Paso, for El Paso; Dr. Hermann S. Rhu, Tucson, and Dr. Preston T. Brown, Phoenix, for Arizona; and Dr. James W. Ravenscroft, San Diego, for San Diego.

The 1959 meeting will be held in Palm Springs, California, November 2 and 3, in El Mirador Hotel.

Speakers Listed

Speakers at the meeting were Dr. Donald L. Hutchinson, Assistant Professor in Obstetrics and Gynecology at the University of California at Los Angeles School of Medicine; Dr. Michael J. Jordan, Associate Professor, Clinical Obstetrics and Gynecology at New York University Medical School; Dr. Ralph A. Reis, Professor of Obstetrics and Gynecology at Northwestern University Medical School; and Dr. Ernest L. Wynder of the Memorial Center for Cancer and Allied Diseases in New York City.

Dr. Charles E. Van Epps, Phoenix, was the retiring President.

Dr. Johnson was born in Elbow Lake, Minnesota, and received his M.D. from the College of Medical Evangelists in Los Angeles. He interned in the Los Angeles County General Hospital and took his residency in the Chicago Lying-In Hospital. He then did a year of postgraduate work in Obstetrics and Gynecology in the Graduate School of the University of Pennsylvania.

Full Commander

He began the practice of medicine in Los Angeles in 1929 and remained there until 1942, when he entered the Navy. Leaving the service as a full Commander following three and one-half years in charge of Obstetrics and Gynecology at the Kern General Hospital in Bakersfield, Cal., Dr. Johnson then practiced medicine in Redwood City, and Sacramento before moving to Santa Ana in 1951.

Dr. Johnson is certified by the American Board of Obstetrics and Gynecology, is a member of the American College of Obstetrics and Gynecology, and was the first President of the Orange County Obstetrical and Gynecological Society. He is a member of the Los Angeles Obstetrical and Gynecological Society, is President of the North Santa Ana Kiwanis Club, and is Chairman of the Advisory Board of the Children's Home Society in Santa Ana. At present he is a member of the California Medical Association's Committee on Maternal and Child Welfare and a member of two state committees of the American College of Obstetrics and Gynecology.

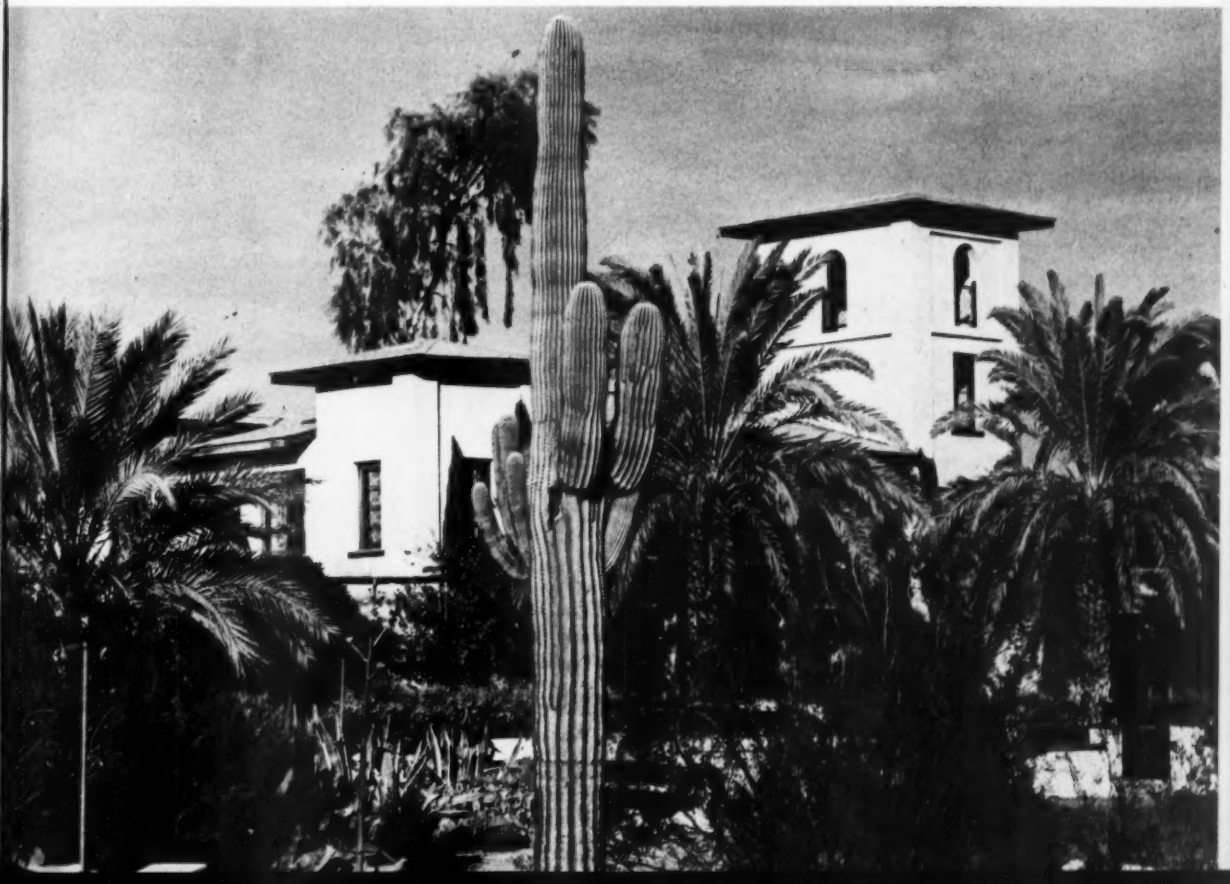
Dr. Johnson's hobby is yachting. He and his wife have a daughter and two grandchildren.

Photographic Report of the

Southwest Obstetrical and Gynecological Society Eighth Annual Meeting, Nov. 14-15, in Phoenix

Above, left to right, are new and retiring officers of the Southwest Obstetrical and Gynecological Society: Dr. Charles E. Van Epps, Phoenix, retiring President; Dr. John F. Wanless, San Diego, Vice-President; Dr. Zeph B. Campbell, Phoenix, Secretary; and Dr. Charles J. Newcomb, Tucson, President-Elect. Not shown are Dr. Donovan Johnson, Santa Ana, President, and Dr. Raymond J. Jennett, Phoenix, Treasurer.

Below, a view of the Paradise Inn, where the meeting was held.



At the round table l
cheon are Dr. Michael
Jordan, left, Associa
Professor, Clinical O
stetrics and Gynecolo
at New York Univers
Medical School, and
Donald L. Hutchins
Assistant Professor in O
stetrics and Gynecolo
at the University of Ca
fornia at Los Angel
School of Medicine. Bo
were guest speakers
the meeting.

Below is an aerial view of the Paradise Inn, where physicians and their wives enjoyed the meeting.

A physician's wife enjoys the Phoenix sunshine.

At the round table luncheon are Dr. Ernest L. Wynder, left, of the Memorial Center for Cancer and Allied Diseases in New York City and Dr. Ralph A. Reis, Professor of Obstetrics and Gynecology at Northwestern Medical School. Both were guest speakers.

To the right is a photo looking down on the part of a spirited round table discussion. Below, Mrs. William C. Johns, left, Albuquerque, and Mrs. Joseph A. Kwint, San Diego, chat in front of the Inn. At the bottom of the page physicians and wives join a group out for a horseback ride.

Dr. Branch Craige New President of El Paso County Medical Society

Dr. Craige

Dr. Branch Craige has been elected President of the El Paso County Medical Society for the 1958-59 year.

Other new officers are Dr. Delphin von Briesen, President-Elect; Dr. Louis W. Breck, Vice-President; Dr. Ward Evans, Secretary; Dr. Bradford Hardie, Secretary-Elect; and Dr. Gilbert Landis, Treasurer. Dr. Gerald H. Jordan is the retiring President.

A native El Pasoan, Dr. Craige is the son of the late Dr. Branch Craige, Sr., well known El Paso pediatrician who practiced in El Paso from 1914 until his death in 1944. His mother was the late Else Kohlberg Craige, the daughter of a pioneer El Paso family. His brother is Dr. Ernest Craige, associate professor of medicine in the University of North Carolina.

AB From North Carolina

Dr. Craige was graduated from El Paso High School in 1931, attended Texas Western College for a year and then the University of North Carolina, where he was graduated with an AB degree in 1935.

He received his M.D. at Harvard Medical School in 1939. He then went to the Peter Bent Brigham Hospital in Boston, where he spent several months in Pathology. He was an Intern, Assistant Resident and Resident at New Haven Hospital, New Haven, Connecticut, from 1940 to 1943 during which he was also on the teaching staff of the Yale Medical School.

He entered the Army in 1943 as a first lieutenant in the Medical Corps of the Army Air Corps. In 1944 he was assigned to the Office of Scientific Research and Development and spent the following two years as army officer in charge of a group investigating the chemotherapy of antimalarial drugs at the University of Chicago, Manteno State Hospital and Stateville Penitentiary in Joliet, Illinois.

Cooperative Investigation

This work was done under the direction of professors at the University of Chicago. Serving in the group were other Army officers, civilian nurses, conscientious objectors and prison inmate volunteers. As a result of this work, which was part of a large cooperative investigation involving scientists in various schools and institutions, Chloroquine and Primaquine, two outstanding antimalarial drugs, were established as safe and effective.

For his share in the study Dr. Craige received the Army Commendation Ribbon and became a member of the National Malarial Society. Not the least of his honors was the much cherished honorary degree of "Ovum Bonum" (good egg) presented by the Stateville Correspondence School, a

school organized and operated by the inmates of the Illinois State Penitentiary.

While in Stateville on the malaria assignment, Dr. Craige met Miss Jean McCracken, who worked in the penitentiary as a secretary. They were married in August, 1945, in Joliet. After he was separated from the service as a Major in 1946, they moved back to El Paso, where Dr. Craige began the practice of diagnosis and internal medicine.

Dr. Craige has been certified by the American Board of Internal Medicine and is a Fellow of the American College of Physicians. He is a member of the Southern Medical Association and the Texas Academy of Internal Medicine.

He is the author or co-author of some 28 articles on medical subjects which have appeared in various medical journals.

Dr. Craige is a former member and President of the El Paso City County Board of Health. He is a Director and Secretary of the Medical Center and a Director and Vice President of the United El Paso Better Health Foundation. He is an Episcopalian and formerly was a vestryman of St. Clement's Church.

He is a member of Sigma Xi, the Boylston Medical Society, Alpha Omega Alpha, Sigma Nu and Phi Beta Kappa.

Dr. and Mrs. Craige live at 2432 Savannah Street and have three children, Betty Jean, 12, a student at Bassett Jr. High School, and Mary Jo, 10, and Branch III, 8, students at Crockett School.

Doctors Have Wives

By JEAN M. CRAIGE, *El Paso*

It is a pleasure to welcome new members and prospective members of our auxiliary, and to start a new year with our old members. One of my good friends made the remark "The Auxiliary exists whether or not we have any meetings." This is certainly true.

We all share similar problems and similar joys, and are more or less automatically given a position of respect in our community. The latter can be a rather frightening responsibility, but I hope to give you a few tips which will make meeting this responsibility a little easier.

"New" Wives

I wonder how many of you "new" wives—women whose husbands have been practicing a short time—have had your non-medical friends ring you up for advice? Medical, of course! They seem to assume that because we're all married to such fine sources of medical information we must use that source constantly.

They don't realize, or can't imagine, that our evenings are not filled with picking our husbands' brains. We should know all about the symptoms and treatment for disseminated lupus, diverticulitis, nephritis, pyelitis, and many other fine-sounding ailments—many of which we can't pronounce!

One of the most difficult chores we have to do is explain tactfully and humorously that our husbands didn't marry us for our medical knowledge! One of my acquaintances once asked me how I became pregnant—and the answer "In the usual way!" didn't seem to quite answer the question.

No Licenses

All of us must remember that we do not have licenses to practice medicine—and must avoid prescribing so much as an aspirin (some people are allergic to it!) We could get into some serious difficulties by doing this.

Then another problem—who would we recommend for such-and-such a symptom? A good answer here is that El Paso is an extremely fortunate community. There are so many well-qualified men in general practice and in every specialty that a decision is really difficult. This is absolutely true. Then you may give the names of four or five men.

There is an exceptionally wonderful spirit of friendliness, comradery, respect and companionship among the members of the medical profession and their wives in El Paso. We are very grateful and thankful for it. Lots of us know this feeling does not exist in many communities. In fact, in one community of which we know—the wives have to switch bridge tallies in order to avoid each other at large parties! In other communities there is not the happy practice of referring patients to each other—some men feel that they prefer to send their patients out of town for care.

Let us all help keep that spirit alive! Let us

learn that when a non-professional friend calls and flies into Dr. So-and-So—(it will happen!) that not only will we give a lip-service defense—but give a sincere defense. Most all of our doctors are working hard to please people twenty-four hours a day. They can't please every one! It's impossible! But if we practice the Golden Rule, our happy, friendly spirit is more apt to remain.

Chief Identity

I sometimes wonder if a doctor's wife is ever judged as an individual! First, we must learn we're always considered "Mrs. Doctor So-and-So." (It takes a little adjusting to learn to accept that fact!) This is our chief identity. Then, of course, we're apt to be a prolific crowd, which is good—and we're Janie's and Billy's mama! We learn to adjust to that. But, after all, what difference does it make who we are, actually? The chief thing to remember along this line is not to do or say anything that could reflect in a bad way on our husbands or our husbands' colleagues.

This brings up the point that we must realize that when we go into the grocery store, the butcher's, the baker's, the candlestick maker's—we're representing our husbands. Let's try to remember that.

One of our hardest problems, which we'll always have with us, is the telephone. If we can learn to be tactful and patient, we will have accomplished something else.

Let's try to do a good job at home—you'll have to assume the great percentage of responsibilities about keeping the home fires burning and keeping the roof from leaking—and keeping the children in line—in order for your husbands to feel free to work as hard as they do.

Doctors' children are very much like ministers' children—cussed and discussed! For goodness' sake, keep them healthy and well-behaved—or papa will suffer (in more ways than one!).

If this sound like a formidable job—I'm sorry. It isn't really! It is a full-time job which requires lots of energy—but what could be more rewarding?

I love to go to a party and meet someone who says "Oh! You're Mrs. Craige. You have a wonderful husband!" My answer to that is "Thank you very much. I think so, too!"

Our Medical Auxiliary meets the first Thursday of every month—sometimes for coffee—sometimes to have lunch. We try to have an interesting and pertinent program.

We have a hospitality committee which tries hard to make new members feel at ease and at home. Please remember, we have become a large group, and it is not easy for us old members to pick up twenty new names at a time. We have badges to help both old and new members get acquainted—we use first names on the badges to foster a feeling of friendliness. (The green ones are for new members, I believe, yellow ones for guests, and white ones for old members).

Old Members

All of us old members remember coming into this group. An old member kindly brought me to my first meeting twelve years ago. Then I started out to attend my second meeting—alone. Halfway here I turned around to go home, feeling I couldn't go into a group of strangers. Then, suddenly, it dawned on me that it would never be any easier to get acquainted. You will become well-acquainted *if* you attend the meetings. If you stay home, it will be more difficult.

If you have a special interest such as decorating, legislation, hospital work, or para-medical recruitment, please tell our President. There are loads of jobs to be done, and volunteers will be welcomed with open arms.

We have an Open House New Year's Day, here at the Turner Home, to view the Sun Parade.

We have a doctors' wives chorus. If you like to sing, make yourself known there.

If your interests lie along the lines of civil defense, safety, publicity, or if you're willing to help with notifications of our meetings—we have many committees where your services will be appreciated.

I recently heard the expression "cultural shock." The incidents of the American ordering beer in England, only to find it warm; the Latin American arriving here with 500 pesos, only to discover the differences in currency; the small town girl going to the city to live. In other words, "cultural shock" is the state of anger at one's self because of ignorance. Let's not come to one meeting, then stay away a year and feel that the group is unfriendly. We get out of the group—or the world—just what we put into it.

Let's remember to be friendly—because this is a friendly group. We're all busy with our own families and our own problems—*But* with a little work—we make new friends easily. We need new members. New members are apt to be new friends.

2432 Savannah Av.

Edited By ANDREW M. BABEY, *M.D.*, LAS CRUCES, N.M.

APHORISMS and MEMORABILIA

Miscellaneous Truths and Concepts

1. "An Esmarch bandage should be restricted to the thigh. There is no place for below-knee tourniquets, and on the upper limb only a pneumatic cuff should be used. A tourniquet should never be put directly on the skin but over toweling. The maximal period of application is two hours, and one hour should rarely be exceeded. A distinct contraindication is in peripheral vascular disease and the elderly, when limb vessels may fracture of thrombose."—Leading Articles, *The Lancet,* May 31, 1958, page 1161.

Hepatic Coma

2. "The possibility of the development of delirium tremens may tempt the clinician to give alcohol to a patient in impending hepatic coma who has recently been drinking heavily. This is rarely necessary and alcohol should be completely withdrawn. The development of delirium tremens is an indication for small doses of serpasil or chlorpromazine." —Hepatic Coma, DR. SHEILA SHERLOCK, *American Journal of Medicine,* May 1958, page 810.

3. "One of the eternal conflicts out of which life is made up is that between the effort of every man to get the most he can for his services, and that of society disguised under the name of capital to get his services for the least possible return."—JUSTICE OLIVER WENDELL HOLMES, *The Holmes Reader,* 1955. p. 279.

4. "No man can go far who never sets down his foot until he knows that the sidewalk is under it." —*Loc. cit.,* p. 282.

Thinking Under Fire

5. "It is one thing to utter a happy phrase from a protected cloister; another to think under fire—to think for action upon, which great interests depend. The most powerful men are apt to go into the melee and fail or come out generals."—*Loc. cit.,* p. 282.

6. "Never do a Master test in a patient who you know full well has functional heart disease merely to satisfy him that he has no organic disease. He may well turn out to have a positive test."—DR. HAROLD D. LEVINE, *Journal A.M.A.,* June 21, 1958, p. 966.

7. "When the clinical diagnosis of acute myocardial infarction is suspect and electrocardiographic support for that diagnosis is yet lacking and no alternative diagnosis has been established, one can have no quarrel with the recording of serial tracings. Sometimes it takes days or even weeks for such changes to appear. But it must be insisted that once a diagnosis of acute infarction has been settled on the basis of dynamic sequential changes there is no good reason for willy-nilly repetition of the electrocardiogram."—*Loc. cit.,* p. 968.

Common Reason

8. "The most common reason given for ordering repeated electrocardiograms is to obtain evidence for extension of infarct. Yet it is only under the most unusual circumstances that the electrocardiogram, as such, gives evidence of this development. The electrocardiogram, in fact, is notoriously inaccurate in demonstrating extension. Much more often than not the clinical story of redevelopment of pain, dyspnea, shock, or sweating is much more helpful in making this inference." *Loc. cit.,* p. 968.

9. "It is particularly noteworthy that the most significant hemodynamic event in clinical medicine, the change from a compensated state to cardiac failure and vice versa, occurs without any known electrocardiographic alterations."—Editorial, *Amer. Journal of Med.,* June, 1958. p. 834.

10. "It is recommended, therefore, that if cardioesophageal tamponage with a balloon tube is necessary to save a patient from exsanguinating hemorrhage, an emergency operation to suture the esophageal varices should be performed without delay."—LINTON *A. Journal of Med.,* June 1958, page 943.

CURRENT THERAPY

Electrolyte Disturbances in the Treatment of Chronic Heart Failure

By M. NATHAN KLEBAN, M.D., *El Paso*

Agents intended for the reduction of edema by augmenting urine formation may produce annoying electrolyte disturbances in the treatment of chronic heart failure. The most practical, effective and widely used drugs interfere with renal tubular ion transport. They bear no direct relationship to implicated factors of elevated renal venous pressure, renal tissue metabolic insufficiency, increased glomerular filtration fraction, or high levels of aldosterone or anti-diuretic hormone. Success is contingent upon the ability of the extravascular fluid to move into the circulation and reach the glomeruli to be filtered into the renal tubule.

Undesirable effects of diuretic drugs are shrinkage of effective circulating blood volume; sodium, potassium or chloride deficiency; and acidosis. They are most likely to occur in a person who is very sick and eating poorly.

Dogged Persistence

Edema and visceral and pulmonary engorgement may fail to yield to dogged persistence in administration of diuretic drugs singly or in combination. Premature systoles may progress to coupled beats, nodal or chaotic rhythm. Subtly and almost imperceptibly or with startling rapidity a mental state of alert interest may shade into dullness, torpor and coma. Desire for food may be lost to be succeeded by distaste and then vomiting. Minor weakness may become profound with muscle discomfort and cramping pain.

When the pathologist examines this person, lying uncomplainingly now in large pieces on a table or in small slices on a microscope stage, there need nowhere be obvious the cause of death. Certain it is that severe heart disease and failure will be found. But the altered balance of water and electrolytes produced by treatment, which worsened the situation or hastened or brought death, is a dynamic state with no incriminating evidence left behind when life has vanished.

Dr. Kleban this month is substituting for Dr. Jack A. Bernard, regular conductor of this section.

Responsibility for rational treatment increases with our expanding fund of available physiological information. The power of our therapeutic weapons generally should be proportional to the danger of the illness we are treating. Jokes which began before recorded history about physicians hastening their patients' departure from life have had a swiftly diminishing factual base with the establishment and expansion of scientific medicine.

Appreciation of the critical function of water and electrolyte balance has brought with it a better understanding of what happens in chronic heart failure, of what treatment does that may be helpful or harmful.

Failure of the heart to meet the work demand thrust upon it means unsatisfied tissue metabolic requirements. Increased pulmonary blood volume imposes greater effort by muscles of ventilation. Expansion of extracellular fluid volume engorges viscera and dependent subcutaneous tissue.

Fundamentals

Fundamentals of long term treatment are to increase the force of the heart beat with a digitalis preparation; to reduce body energy demands by restricting activity; preventing or correcting such stress as infection, endocrine disorder or emotional turbulence; and to contract swollen extracellular fluid space by sodium frugality and warring against the factors operating within the kidney tubular cells responsible for salt and water reabsorption.

Great mischief flows from this internal sea water flood and may be compounded by desperate measures to reverse the flow to an ebb tide.

Contraction of effective circulating intravascular fluid volume may result from urine formation proceeding at a faster rate than movement of edema back into the circulation. This may happen when fluid is being sequestered into a

body cavity such as the pleural space, or when there is saphenous venous system insufficiency.

Massive Diuresis

This will occur with massive diuresis or may occur with only moderate diuresis in a person with chronic edema. Signs are hypotension and tachycardia, especially in the upright position, leading to shock and those signs which indicate cardiac, brain and other organ anoxemia. Edema may be present in the dependent parts of the body, but the skin of the arms and face will be sticky, shrivelled and inelastic, the tongue longitudinally wrinkled, the eyeballs sunken-signs of extracelluar salt water depletion.

This grave state of affairs may be prevented by discrete use of diuretics, by looking for and removing pleural fluid by aspiration if present before using diuretics, and by wrapping elastic bandages around the legs if there is cronic edema with or without venous insufficiency at the time a diuretic is given. Cautious administration of intra-venous balanced electrolyte solution would have to be given if signs of shock were present.

Common Disorder

Probably the most common electrolyte disorder induced by the doctor is potassium depletion. The effective diuretic drugs disrupt renal tubular cell electrolyte transport systems but not in any controllable selective fashion, nor do they have any direct effect on water reabsorption.

Urine formation is enhanced by the osmotic effect of tubular ions. It is not known how many renal tubular cell electrolyte transport systems there are. Definite information about mode of action of diuretics in the tubule cell is available for only one group, revealed by its name, carbonic anhydrase inhibitor.

Mercurials, carbonic anhydrase inhibitors and chlorothiazide, an organic heterocyclic sulfur compound trade marked Diuril, sweep out potassium indiscriminately along with sodium and chloride. There is some, but not conclusive, evidence that mercurials interfere primarily with chloride transport and that they probably react with an —SH (sulfydryl) enzyme system, such as succinic dehydrogenase.

Diuril, second only to parenteral mercurial in potency and at times effective when response to mercurial has been lost, is more likely to cause trouble from potassium loss. Serum potassium levels tend to fall with a total daily dose of 2.0 grams or more of Diuril.

Muscle Mass Lost

People in severe chronic heart failure usually lose muscle mass. Breakdown of tissue is accompanied by negative nitrogen, potassium and phosphate balance. Hypokalemia should be especially watched for if food intake is poor (which means poor potassium ingestion) or if corticosteroids are being given.

Flaccid muscular weakness and ileus are prominent clinical signs. Since potassium ions leave cells in negative balance serum potassium determination will not necessarily be below the lower limit of and may be above the normal range of 4-6 mEq/L on any single determination.

And since the effects of potassium balance are partially dependent on intra to extracellular ratio as well as other cations, the ECG changes of ST segment depression, flattening or inversion of the T wave and lengthening of the Q-T interval may not necessarily be demonstrable on any single tracing.

Metabolic alkalosis usually accompanies negative potassium balance. For each three potassium ions lost by the cell, it gains two sodium and one hydrogen ion. Extracellular hydrogen ion concentration drops and intracellular hydrogen ion concentration rises. Because the kidney tubule cells are also affected by the rise of H+, compensatory H+ conservation does not occur.

Corrects Balance

Potassium corrects negative potassium balance with or without alkalosis. One to 6 grams potassium should be given daily when Diuril dosage is 2.0 grams or more per day. Potassium is available in 0.3 and 0.5 gram tablets as the chloride salt and as a solution of acetate, carbonate and citrate each in 10 per cent concentration supplying about 15 mEq of potassium per 5 ml. It is most palatable diluted in fruit juice. A 40 mEq ampul diluted in 1000 ml. of fluid may be given safely intra-venously when there is urine formation of about 50 ml./hour or more.

The arrythmias of digitalis intoxication, such as premature systoles, atrial, a-v nodal or ven-

tricular tachycardia, artrial fibrillation or chaotic rhythm may actually be due to potassium depletion. Potassium balance should be determined before concluding that glycoside poisoning is present.

Failure to Respond

Failure to respond to mercurial diuretics may be due to hypochloremic alkalosis. Mercurial diuresis yields almost equivalent chloride and sodium loss. Since the extracellular concentration ratio of chloride to sodium is 1:1.4 there is a relatively greater loss of chloride.

Osmolarity is maintained by an increase in bicarbonate anion. This shifts the base bicarbonate to carbonic acid of 20:1 in favor of base bicarbonate, raising the pH. Diminished respiratory minute volume conserves carbon dioxide, increasing carbonic acid, and reestablishing a neutral pH.

Ammonium chloride corrects this defect. As a diuretic this salt has little value alone, but it enhances the potency of mercurials. There is strong disagreement whether it accomplishes this by lowering pH or by furnishing chloride ion to the glomerular filtrate. Ammonium is methabolized to urea.

If given to potentiate a mercurial diuretic the dose is between 8 and 12 grams a day given the day before and the day of the mercurial injection. There is no value in giving ammonium chloride for any longer period of time because the renal response of its own mechanism of elaborating NH4 in defense of acid-base neutrality reaches a peak by the third day. By this time NH4Cl is without effect and should not be given again for several days.

Lower Serum Sodium

Low serum sodium may reflect an intracellular distributional shift, dilution due to water retained in excess of sodium, or a total body sodium depletion. Patients with chronic congestive heart failure on salt restriction, cation exchange resins or diuretics may be asymptomatic and edema-free but have hyponatremia resistant to correction. In other reported cases sodium levels returned to normal with cessation of therapy. Presumably the sodium in this situation shifts into the intracellular compartment.

The real low sodium problems are severely decompensated, edematous, hypochloremic, and azotemic. They are either on 200 mg. sodium diets or are practically not eating. They are treated vigorously and desperately but continue to deteriorate. Meticulous search should be made for infection, pulmonary infarction, active rheumatic myocarditis, potassium deficiency and underdigitalization which may be masked by hypokalemia. Control or correction of these factors may boost sodium back to normal tonicity.

No Single Factor

No single factor operates to produce hyponatremia. Exchange resins such as Carbacrylamine resins are a mixture of cation exchange resins in the hydrogen and potassium cycle and anion exchange resins. The minimum dose of 16 grams taken three times a day between meals in water or other liquid or mixed with food is a practical barrier to its use. Mild acidosis is not usually a problem. Supplementary calcium salt is prescribed at bedtime to correct hypocalcemia when resins are taken over a long period of time.

Mercurial diuretics alone cannot produce hyponatremia, becaues they cannot remove sodium in excess of water; diuresis from mercurials contains sodium in concentration below serum levels. Mercury in the form of Calomel (mercurous chloride) was used as a diuretic 400 years ago. The first organic mercurial was used to treat syphilis with poor results but it was found to be a powerful diuretic.

The most commonly used preparations are Mercuhydrin (meralluride), Salyrgan-Theophylline (mersalyl and theophylline), and Thiomerin (mercaptomerin). Each ml. contains the equivalent of about 40 mg. of mercury. Dosage is 1-2 ml. There is no advantage to the i-v route unless circulation through the deep muscles is so poor that the drug may not be absorbed.

It is said by wise and experienced cardiologists that if one persists in the intravenous administration of mercurials over a long enough period that one will encounter a fatal reaction.

Combination of Factors

The combination of sparse sodium intake, diuretics, and other factors generally operate, then, to yield the malignant low salt syndrome.

Water in excess of sodium retention is attributed to sustained high levels of antidiuretic hormone stimulated by an extraosmoreceptor mechanism. The theory lacks proof.

If one can find no contributory correctible factors then one gives hypertonic sodium chloride in 2.5 or 5% concentration. The amount may be calculated on the basis of the difference between a normal serum sodium value of 140 mEq and the patient's value multiplied by either extracellular fluid volume or total body water.

There is disagreement on this last point. And the disagreement is a pretty big one. Or one can simply give about 250 ml. at a time, slowly and with prayer, and check the serum sodium before giving more. Of course water must be withheld. If it isn't the salt will simply be diluted.

This situation is rather discouraging. The serum sodium may be raised slightly, but the pulmonary edema probably will also. And one can conclude at this point that an unbridgeable gap has been reached between the ability of the heart to perform work and the tissue metabolic demands.

Diamox and Cardrase

Diamox (acetazolamide) and Cardrase (ethoxzolamide) are carbonic anhydrase inhibitors developed from the observation that sulfanilamide produced metabolic acidosis. Carbonic anhydrase is an enzyme found in red blood cells, gastric mucosa, renal tubule cells and elsewhere.

It catalyzes the reaction $CO_2 + H_2O - H + HCO_3$. Around this reaction revolves the function of the renal tubule in maintaining a neutral pH. H+ is made available for exchange with tubular sodium or for elaboration of NH_4+. Sodium, and to a lesser extent other cations, is excreted in the urine in increased amount while H+ is conserved.

This may lead to metabolic acidosis. For this reason Diamox is given in a dose of 250 to 500 mg. and Cardrase from 62.5 mg. every other day or on similar alternating schedules.

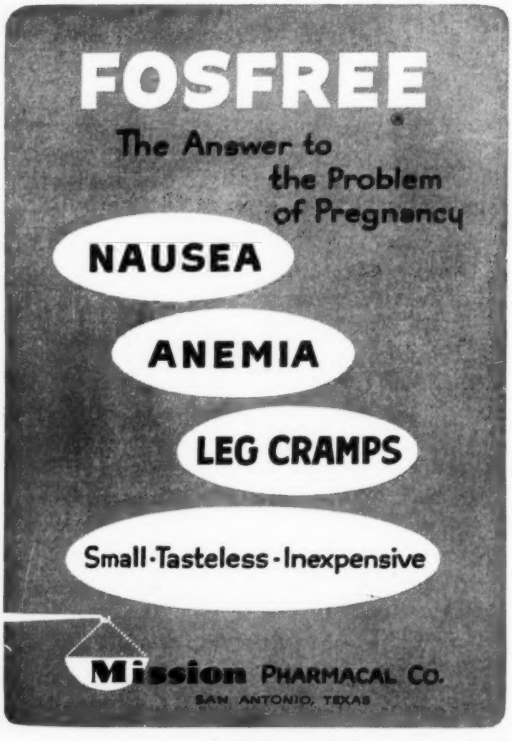
Carbonic anhydrase inhibitors used on an alternating schedule with Diuril or mercurials tend to balance out acid-base derangements, but still deplete potassium stores. Diamox or Cardrase seem to benefit patients who have heart disease secondary to or associated with chronic bronchopulmonary disease and carbon dioxide retention.

It is paradoxical that an agent which cancels compensatory sodium bicarbonate elevation response to respiratory acidosis by induction of metabolic acidosis should be beneficial rather than harmful.

Aminophylline is useful as a diuretic in combination with a mercurial. Two or 3 hours after the mercurial is injected 0.5 gm. of aminophylline is given slowly intravenously. It is timed to coincide with the peak effect of the mercurial. Enhancement of diuresis results from the increased glomerular filtrate which in turn probably is brought about by increased cardiac output and renal blood flow.

Aminouracils, Mictine and Rolicton, are moderately effective diuretics which produce no significant electrolyte disturbances.

Distinction between the inevitable end of decompensated heart disease and a desperate, superficially hopeless but correctible dislocation of fluid volume and electrolyte concentration and composition brought about partially or entirely by treatment is difficult but possible. Use of available physiological and pharmacological information, adequate history, astute physical examination and dependable laboratory determinations are required for optimum clinical evaluation.



MONTHLY CLINICAL PATHOLOGICAL CONFERENCE
EL PASO GENERAL HOSPITAL

October 16, 1958

FREDERICK P. BORNSTEIN, M.D.—*Editor*—*Case No. 931*
Presentation of case by Dr. William R. Gaddis,

Introductory Note:

Due to a break-down in recording equipment this conference is presented in abbreviated form.

History: Dr. Nathan Kleban:

Abdominal pain brought a 35-year-old unemployed single Latin-American man to the hospital on October 2, 1957. Chills and generalized abdominal pain began five days before admission. The patient experienced diffiulty in urinating, chills stopped, pain persisted, and vomiting of clear fluid occured before admission.

Past history was not remarkable. The father had died of some sort of heart trouble. The mother was in good health. There were no siblings.

Physical Examination

The patient was described as being mentally disturbed. T. 102. P. 112. R. 22. Blood pressure was not recorded. Oral hygiene was poor. Breath sounds were diminished at both lung bases. Abdominal tenderness was diffuse.

Hospital Coure:

Treatment consisted of parenteral fluids, sulfisoxazole Gantrisin, and meperidine (Demarol) for pain. Temperature rose to 103 but dropped to 98.2 the following day when he seemed to be confused, had a large, soft stool in bed and four in the commode.

Maximum temperature on the third day was 100. After being transferred to the medicine service he was given Sparine for nausea. It was noted that he carried his passport to the bathroom.

Acute abdomen was the preoperative diagnosis when the patient was subjected to surgical exploration on the fourth day. Generalized purulent peritonitis was encountered. The gangrenous retroceal appendix was removed.

After surgery the patient gagged and had trouble breathing. (Oxygen was administered then). Vasopressor agents were given for hypotension. Rectal temperature rose to 106.

Shallow, Rapid Respirations

Respirations were shallow, rapid, with suprasternal retraction and crepitant rales on the fifth hospital day. The patient was restless. Rectal temperature remained 106. The abdomen was soft. Chloramphenicol, tetracycline, hydrocortisone, gamma globulin and parenteral fluids were given. Bronchi were aspirated and oxygen with Alevaire delivered through a tracheotomy. Blood pressure stabilized and sensorium cleared for a few hours. Urine appeared to contain bilirubin.

Jaundice was noted on the sixth day. Sensorium became cloudier. Rectal temperature dropped to 102. There was bleeding from the tracheotomy wound.

Respirations became gasping and more labored. Uremic frost was seen on the patient's face. Coma succeeded stupor. Blood pressure could not be maintained with vasopressors. Death occurred on the seventh hospital and third post-operative day.

Laboratory Findings

X-ray: 10-4-57—Abdomen: "Survey film of the abdomen reveals no evidence of free air under either hemidiaphragm. The kidneys are normal in size, shape and position. The psoas shadows and properitoneal fat lines are present bilaterally. A marked amount of gas is present in the stomach and colon. The lumbar spine and pelvis appear natural. Conclusion: Abdomen negative for evidence of mechanical obstruction of opaque calculi."

10-6-58—Chest (portable). "Portable examination of the chest reveals bilateral confluences suggestive of a possible tracheo-bronchitis. There is a tracheotomy tube lying within the trachea. There is bilateral subcutaneous emphysema in the region of the neck. Accompanying rib fractures cannot be entirely excluded. Conclusion: Subcutaneous emphysema. Possible fractured ribs."

Blood counts:

10-2-57: Hb. 13. 8 gms., Ht. 50 vol.%, WBC 5,500, Stabs 5, Segs. 69, Lymphs. 24, Monos. 2.

10-2-57: Hb. 13.3 gms., Ht. 49%, WBC 6,400, Stabs. 17, Segs. 44, Lymphs. 39.

10-4-57: Hb. 13.1 gms., Ht. 45%, WBC 5,800, Segs. 78, Lymphs. 22.

10-5-57: Hb. 13. 8 gms., Ht. 46%, WBC 12,700, Stabs. 5, Segs. 84, Lymphs. 8, Monos. 3.

10-6-57: Hb. 12.9 gms., Ht. 50 Vol.%.
10-6-57: Hb. 14.2 gms., Ht. 53 Vol.%.
10-7-57: Hb. 12.4 gms., Ht. 39%, WBC 28,900, Stabs. 6, Segs. 89, Lymphs. 1, Monos. 4.

Blood Chemistry:

10-5-57: Urea Nitrogen—27.5 gm.%.
10-7-57: CO_2 Capacity—12.9 mEq/L.
Chlorides—445.
Nonprotein Nitrogen—130 mg.T.
Potassium—6.1 mEq/L.
Sodium—108 mEq/L.
Van den Bergh Direct—3.5 mg.%
Indirect—5.0 mg.%
C1—124.5 mEq/L.

Urinalysis:

10-2-57: (Catheterized)—Yellow, cloudy, acid, S.G. 1.017, Albumin 4+, Sugar negative, 1-3 WBC, 0-2 RBC, rare Ep. cell, many coarsely granular casts.
10-3-57: Yellow, cloudy, alkaline, S.C. 1.020, sugar and albumin negative, microscopic negative, urate crystals.
10-5-57: "Very deep yellow", very turbid, reaction 5.5, S.G. 1.012, Albumin 2+, Sugar negative, WBC 10-15/HPF, few Eq. cells, occasional fatty casts, abundant bacteria, few urate crystals.
10-6-57: Negative for bile.

Bacteriology:

Smear and culture, pus from abdomen—E. coli —sensitivity Choloromycetin and Tetracycline.

Serology:

10:9-57: Negative.

X-Ray Discussion: Dr. William H. Melton:

The upright and flat films show the distended stomach which contains a considerable amount of gas and also some gas in what appears to be the transverse colon. I can't see the diaphragms on the upright examination, so I cannot exclude the presence of free air.

I do see a properitoneal fat line which in the presence of purulent peritonitis you wouldn't expect to see. The other soft tissue structures are not well defined. I see some shadows on the flat film that look like some gas in the small bowel but the gut pattern is not really diagnostic.

More Consistent

It is more consistent I think with a moderate degree of adynamic ileus than a mechanical obstruction and the abdonimal films all in all don't tell you a whole lot. The chest film is a portable examination and detail is poor on it. A tracheotomy tube is in place and a considerable amount of subcutaneous emphysema is present, primarily of the neck regions and I think I can see some mediastinal air on both sides in the superior mediastinum, in addition to some irregular density in the right chest which has the appearance of an inflammatory change.

Summary

In summary, the patient has a tracheotomy with bilateral subcutaneous emphysema, possibly some mediastinal emphysema, and what appears to be an inflammatory infiltration in the right lung field. On the abdominal film the findings are consistent with a moderate degree of adynamic ileus. I cannot tell if there is free air.

Dr. Gaddis:

The problem with this patient was one of chills, fever and abdominal pain. There is very little to to discuss about the history. I thought that it would be easier to discuss this case from the standpoint of various chart notations that were made regarding his history, physical and hospital course and laboratory findings.

First, let us say his habits were unknown prior to his illness. We infer that he crossed the border as an alien; it was noted that he carried his passport on one occasion. In a discussion of the physical examination, he was "mentally disturbed". This can mean many things; agitation, depression, disorientation, hallucinations, delusions, etc. What we don't know about this patient is manifold and precludes a long discussion.

It might be interesting to wonder if the symptoms and findings could be compatible with drug withdrawal. In a discussion of his hospital course, this patient exhibited intermittent high fever which is evidence of sepsis. He had the abdominal findings of appendicitis and peritonitis. In addition we encounter cerebral depression, acidosis, coma and death with urinary suppression and uremia.

Laboratory Findings

A discussion of the laboratory findings: First of all the blood counts show the progression to the leukemoid reaction as a result of the severe infection. The chemistries are inadequate. I have acidosis, mineral deficiency, renal inpairment and liver involvement.

Diagnostic impression: The onset of this disease is in line with acute hepatitis with a superimposed acute appendicitis. It should be assumed then that the primary disease was acute appendicitis following which we have a pylephlebitis, hepatic coma and death. This is added to a kidney previously diseased from a latent glomerulonephritis.

Clinical Diagnosis: Acute peritonitis.

Dr. Gaddis' Diagnosis: Pylephlebitis, hepatic coma and death.

Pathological Diagnosis: Pylephlebitis with liver abscess.

Pathological Discussion—Dr. Bornstein:

On autopsy we found the body of a slender young man with a freshly sutured appendectomy wound. The main pathological findings were present in the peritoneal cavity. The peritoneum was dull, hyperemic, jaundiced and covered with thick flakes of fibrin.

The intestinal loops were matted together by these flakes of fibrin. Large pools of pus were present throughout the peritoneal cavity. At the site of operation the wound was closed and intact. However, arising from the region of the ileo-cecal valve one could palpate a thick, indurated, hyperemic strand of tissue which on opening shows a vein with a necrotic intima. This vein was partially obstructed by a thrombus. (Fig. 1).

Figure 1

This thrombotic lesion could be traced into the portal vein and from there into the liver. The liver weighed 2100 grams. The surface of the liver was dull and covered with fibrin. On sectioning one saw several wedge-shaped, sharply demarcated areas in the right lobe which measured up to 8 cm. in greatest diameter. (Fig. 2).

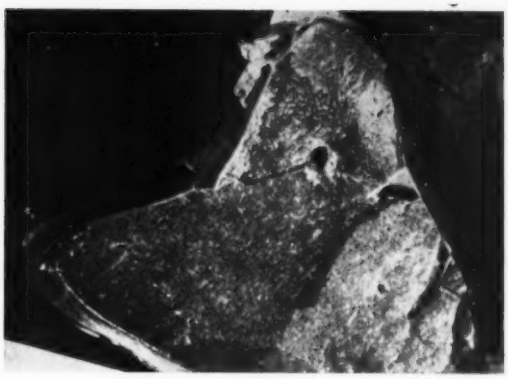


Figure 2

These lobes were supplied by the thrombotic vein mentioned before. On microscopic examination one saw extreme necrosis of liver tissue in areas adjacent to these thrombosed veins, with a picture of a typical thrombophlebitis. (Fig. 3).

Figure 3

Well Known Process

The process we observe here is a well known one, although it has become exceedingly rare at the present time. It is obvious that this process started with a perforated appendix. However, at the time the appendix was removed, a secondary focus of infection had established itself in the appendiceal region.

Beginning from here the process had extended through a mesenteric vein, producing ascending thrombophlebitis which finally extended into the portal vein and liver. This, then, is the classical picture of sepsis.

The picture of sepsis occurs when a focus of inflammation is established in the body which, for

example, originates in the skin (Staphylococcus or streptococcus), or in a lymph node (typhoid fever), or in the lung (miliary tuberculosis). This pathogenic organism may then settle in a location such as an abcess or a vein where there is constant communication with the circulatory system.

Infectious Agents Released

At regular or irregular intervals active infectious agents are released into the circulation where they are usually destroyed to a large extent, an event commonly associated with a chill. The remainder will colonize and produce septic metastases in other organs which again may produce new septic foci.

The blood stream itself is rarely the active seat of the infection and only as a terminal event does the infectious agent multiply while being carried along the blood stream. Sepsis essentially is a process of local growth of infectious agents.

Problems of this sort have become rare and unusual; however, I am afraid that with the progressive development of strains of pathogenic organisms resistant to antibiotics, the problem will become more common and more serious in the next few years.

Some Thoughts About Clinical-Pathological-Conferences

From a beginning when there were Pathologists and Clinicians getting together debating around the autopsy table, the present Clinical Pathological Conference has developed until it has now become a standardized function with motions as stately and prescribed as those of a French minuet. Everything about these conferences has been solidified by tradition, and they have developed their own rituals and unfortunately with them certain preconceived notions. The time has come to ask whether this ritual is worth the time of innumerable physicians all over the country.

It is not the purpose of a Clinical-Pathological-Conference to be a spectacle like a bull-fight, depending upon one's point of view whether the Clinician or the Pathologist is a baited victim or the one who delivers the final death stroke. Nor is it the purpose of the conference to make the obvious obscure and the obscure obvious.

To some extent the character of the conference will be shaped by the subjective outlook of the man whose responsibility it is to pick the material. In selecting the material I have certain specific goals in mind for such a conference.

The first and most important one remains proper correlation between what went on during the disease and what we find post mortem; and, while I believe that the autopsy is the most important part of the post mortem examination, the entire Pathological picture, including the disturbed chemical and physiological dynamics, have to be part of this total correlation.

In short, these meetings should aid in a better understanding of the nature of the disease process and should furnish new approaches to more accurate diagnosis. Therefore, tricks and red herrings have no business in a Clinical Pathological Conference. The famous suppression of facts often imputed to the Pathologist destroys the aims of the whole procedure.

Vivid Memory

Secondly, I do not like to use canned and old cases. The more vividly the individual patient remains in the memory of the staff and the more vivid the personal experience of the clinical problem remains with the staff members, the more value the conference will have.

If such cases sometimes are rare and unusual, so much the better. It emphasizes that diagnosis cannot be made by statistics and that the rare and unusual is always with us. We should not forget that the rare and unusual of yesterday may become the common place of today, and vice versa.

It would have been hard to believe 50 years ago that erysipelas would become less common than lupus erythematodes or pulmonary coccidioidomycosis more common than lobar pneumonia.

The Clinical Pathological Conference is a place where we try to reduce the ever shifting and variable phenomena of the living substance to a rational base (this base being Pathology in the widest sense), with the intention of increasing clinical understanding not as a duel but as a cooperative effort. With this in mind I believe that the Clinical Pathological Conference has a useful function to perform.

FPB, 616 Mills Building